119, 245). These abnormalities prevailed in one patient during anuria after diabetic acidosis; in one example of Chiari's syndrome after operation to produce portacaval shunt followed by numerous taps of the peritoneum and pleura; and in 14 patients with the nephrotic syndrome.

Correction of low plasma sodium and bicarbonate might be hoped to augment plasma volume and subsequently increase output of urine and chloride, thereby removing anasarca. Accordingly, sodium lactate and subsequently sodium and potassium acetate were administered orally. Initially body weight increased. Plasma sodiums rose from 120-135 mEq per liter to above normal; plasma bicarbonates from 9-20 mEq per liter to normal or above. Plasma volumes estimated from falling hematocrit expanded as much as 40 per cent. Daily clearances of sodium, bicarbonate and endogenous creatinine increased several fold. Daily urine flow rose from 0.05 to over 4.0 cc. per minute as urine chloride concentration increased from 0.1 to 1.5 times the plasma value. Anasarca then disappeared.

In the balance studies the recovery of sodium approximated that anticipated from the volume of edema fluid eliminated but the chloride jettisoned was in marked excess.

Although there are other procedures for the relief of anuria (artificial kidney, peritoneal lavage) the relative simplicity of correcting the biochemical and physiologic abnormalities in anuria suggests that this procedure receive more extensive trial. Likewise in the nephrotic syndrome, the results to date warrant further study of the significance of the electrolyte abnormalities.

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*Evaluation of Pentaquine as a Cure of Relapsing Vivax Malaria**

A Controlled Study of Ninety-five Cases

BERNARD STRAUS and JOSEPH GENNIS

Investigations reported by the Board for the Coordination of Malarial Studies indicated that pentaquine (SN 13,276) 6-methoxy - 8 - (5 - isopropylaminoamylamino) - quinoline, when combined with quinine is effective in the eradication of relapsing P. vivax malaria.

The present investigation was designed to test the efficacy of pentaquine when combined with quinine in the definitive therapy of the naturally acquired P. vivax infection in man.

Methods: The patients studied were all veterans of World War II who were hospitalized at the Veterans Administration Hospital, Bronx, New York. Because of progressive development of immunity and the unpredictability of the relapse rate it was decided to employ a control series. Chloroquine (SN 7618), 7-chloro-4 (4-diethylamino - 1 - methylbutylamino) quinoline, was selected for the control series because it is a highly effective agent in the treatment of the immediate attack and has little, if any, effect on the relapse rate. Chloroquine diphosphate was administered in a dosage of 0.6 gm. of chloroquine base followed in six hours by 0.3 grams and 0.3 grams on the second and third days. Pentaquine was employed as the mono-phosphate in a daily dose of 30 mgms of pentaquine base, 10 mgms being given every 8 hours together with 0.6 grams of quinine sulphate. This regimen was maintained for 14 days. Pentaquine was given in one-half of the recommended daily dosage of 60 mgms of base in an attempt to reduce toxicity. This was feasible because there was reason to believe that some degree of immunity had been developed by most of the veterans who would undergo therapy.

Beginning in January 1947, alternate

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cases were selected for either chloroquine or pentaquine therapy. All patients had proven vivax malaria.

There were 46 patients in the chloroquine series. Forty of these patients saw service in the Southwest Pacific area. There were 49 cases in the pentaquine group. Forty-four of these served in the Southwest Pacific area.

Follow up studies were made at approximately monthly intervals. The follow up study is still under way. The length of follow up varies from 1 week to 14 months. All patients will be followed for a minimum period of one year. Eighty-five per cent of the entire group have had a follow up period of more than 4 months at this time.

Results: None of the 49 cases in the pentaquine group has relapsed to date. In the chloroquine control group of 46 patients, there were relapses in 14 patients or 30 per cent. Twelve of these were from the Southwest Pacific. There were 18 relapses in the 14 patients. One patient had three relapses; two patients suffered two relapses; 11 patients have had one relapse. Four patients in the chloroquine group were subsequently treated in a relapse with pentaquine and are also included in the pentaquine series. All other chloroquine relapses were treated subsequently with chloroquine.

Toxicity: Toxic manifestations to pentaquine-quinine were minor. In no case was it necessary to discontinue pentaquine because of toxicity. In one case it was necessary to discontinue quinine. Some toxic manifestations were observed in 75 per cent of patients. Most of these were insignificant. Nausea and anorexia were common during the first four days. Vomiting occasionally occurred during this period. Mild abdominal pain was relatively frequent after the first week. Tinnitus, dizziness and headache were not infrequent. No frank hemolytic reactions were observed, but 6 patients showed a drop of 1 million red blood cells or less per cu. mm. of blood in the course of therapy. Drug fever of 99.6-103.4 degrees developed in 7 patients, from the 7th to 11th day of therapy, and subsided spontaneously in one day. Many of the toxic manifestations probably were due to quinine.

Conclusion: In the treatment of relapsing P. vivax malaria of World War II veterans, pentaquine-quinine in the indicated dosage is apparently a highly effective curative agent. Toxic manifestations were insignificant at the dosage level employed. These results confirm the observations of Alving and of Coatney that even with one-half the previously recommended dosage of pentaquine, eradication of relapsing vivax malaria is achieved. Further follow-up is essential before a final evaluation can be made.

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Differential Diagnosis of Diaphragmatic Hernia and Coronary Heart Disease

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It has frequently been observed that patients with diaphragmatic hernia have pain in the lower part of the chest; in coronary artery disease, too, gastrointestinal symptoms are common. Thus the differential diagnosis of hiatus hernia and disease of the coronary artery is important. During the past twenty years, there has been a marked increase in the frequency of occurrence of both conditions. On the basis of the physiological experiments of Dietrich and Schwiegk and of Gilbert and Fenn, it has been assumed that diaphragmatic hernia produces constriction of the coronary artery reflexly through the vagus nerve, and that the two conditions are interdependent.

We are of the opinion that hiatus hernia can be distinguished from coronary artery